Definitive Embolization of Meningiomas
A Review

B.T. BATEMAN, E. LIN*, J. PILE-SPELLMAN**

College of Physicians and Surgeons, Columbia University
**Department of Radiology Columbia University*
***Department of Radiology, Columbia University*

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Summary

This review examines the possible role for definitive embolization as a primary therapy for intracranial meningiomas. Surgery or radiosurgery are currently considered the standard of care for most benign meningiomas. However, each of these carries substantial risks. The perioperative mortality for surgical resection, as reported in large series, is between 3.7-9.4%; these studies report a similarly high rate of new neurological deficits following surgery. The rate of complications from radiosurgery is reported between 2-16% and it may take months to years before improvement in symptoms occurs following this therapy.

There are a few reports of treating meningiomas by embolization without subsequent surgery. While these studies include small numbers of patients and have limited follow-up, the initial results are very promising.

Given the risks and limitations of surgery and radiosurgery, prospective trials are now needed to determine the safety and efficacy of definitive embolization.

Background

Meningiomas are the second most common type of intracranial tumor, accounting for 13-26% of all primary intracranial tumors [1]. The incidence of symptomatic meningiomas is estimated to be 2 per 100,000 person-years [2]. Approximately 90% of meningiomas are benign, 6% are atypical, and 2% are anaplastic [3]; only 0.2% of meningiomas metastasize. They usually come to clinical attention when the tumor compresses adjacent structures, such as brain tissue, CSF pathways, cortical veins, and major venous sinuses, causing signs and symptoms which may include seizure, headache, or focal neurological deficits [4].

The standard therapies for meningiomas carry a high risk of morbidity and morality, and are not fully efficacious. This, coupled with recent reports of successful treatment of meningiomas with definitive embolization, suggests the possibility of a role for intra-arterial embolization as a primary therapy for this disease.

In this article, we will discuss the standard therapies for meningiomas and the associated outcomes, the results from the few studies published on definitive embolization, and finally some of the risks and the possible benefits of treating meningiomas in this manner. It is hoped that this review may provide an impetus for clinical trials examining the safety and efficacy of definitive meningioma embolization.

Problems with standard therapies for meningiomas.

The management of a patient with a meningioma is dependant on multiple factors including the patient's symptomology, the size and site of the tumor, and the patient's general

health [4]. Patients who have small, asymptomatic meningiomas that are discovered incidentally on neuroimaging can be reasonably managed conservatively, with close follow-up for clinical and/or radiographic signs of tumor progression [4-6]. For symptomatic patients who have surgically accessible tumors, and whose general health permits the risks of surgery, the primary treatment is generally an attempt at complete resection of the tumor [3,4,7]. Radiotherapy has been shown to have a role as an adjunct to surgery in patients in whom surgical resection was incomplete [8,9], in patients with recurrent meningiomas [10], and in patients with atypical or anaplastic meningiomas [11-13]. Further, mounting evidence suggests that efficacy of radiosurgery as the primary therapy for some meningiomas.

Surgical outcomes

The risks associated with surgical resection of meningiomas are substantial. The most commonly encountered post-operative medical complications, which are often related to new neurological deficits created during surgery, include deep vein thrombosis, pulmonary embolism, myocardial infarction, arrhythmia, and pneumonia [14,15]. These complications result in a relatively high peri-operative mortality rate. The largest case series published in the last 15 years had the following results: Kallio et Al., in a series of 935 patients, found a peri-operative mortality rate of 9.4% [16]; Meixensberger et Al, in a series of 385 patients, found a peri-operative mortality rate of 4.2% [17]; Altinors et Al., in a series of 344 patients, found a peri-operative mortality rate of 5.8% [18]; and Stafford et Al., in a series of 581 patients, found a peri-operative mortality rate of 3.7% [19]. In elderly patients, the peri-operative mortality rates have tended to be even higher. A review of case series published during the last 15 years involving patients over 65 shows peri-operative mortality rates ranging from 1.8%-16% [15,20-23].

New neurological deficits, which in many instances are permanent and severely disabling, are a major source of morbidity following surgical resection of meningiomas. Cortical deficits can result when the plane between the arachnoid and pia is adherent to the tumor, such that when the tumor is resected, pial vasculature is destroyed and microinfarction results [24]. Cranial neuropathies are a significant problem following the resection of skull base and posterior fossa tumors [4,24-27]. The rate of new or worsened neurological dysfunction following meningioma resection has been reported at approximately 10% in several large series involving tumors in multiple intracranial locations [15,18].

The rate of new or worsened dysfunction is much higher for tumors in some locations. Roberti et Al. reported a 41% rate of permanent neurological deficit following surgery in a series of 161 posterior fossa meningiomas [27]. In their series of 39 patients who underwent resection of meningiomas of the cavernous sinus, O'Sullivan et Al. reported a 17.9% rate of new dysfunction for cranial nerves controlling extraoccular movement, an 8% rate of new trigeminal nerve dysfunction, and a 12.8% rate of permanent hemiparesis [26]. Zentner et Al., in their series of 19 patients operated on for petroclival meningiomas, reported an 11% rate of new major permanent post-operative neurological deficits [25].

The post-operative deficits experienced by some patients are reflected in studies that assess quality of life following meningioma surgery. In their survey of 155 patients who underwent meningioma resection, Kalkanis et Al. report that 23% of respondents gave a negative or equivocal answer to the question "Are you content with the quality of your life?" [28]. Mohsenipour et Al., in their survey of 82 patients, found that 39% reported a moderate or severe impairment of their quality of life following meningioma surgery [29].

It should also be noted that surgery is not a completely effective therapy for meningiomas. In Jaaskelainen's analysis of 657 patients who underwent surgical resection of benign meningiomas, the estimated recurrence rate was 19% at 20 years [30]. Mirimanoff et Al. followed 225 patients after meningioma resection, finding the recurrence rate at 5, 10, and 15 years to be 7%, 20%, and 32% respectively following complete resection, and 37%, 55%, and 91% following subtotal resection.

The probability of having a second operation by year 15 following complete tumor resection was 20%, and 84% following subtotal resection in this group [31]. Kallio et Al., who followed 935 patients after surgery, found an increased mortality rate relative to the general population of 9% at three months, 11% at one year, and 22% at 15 years [16].

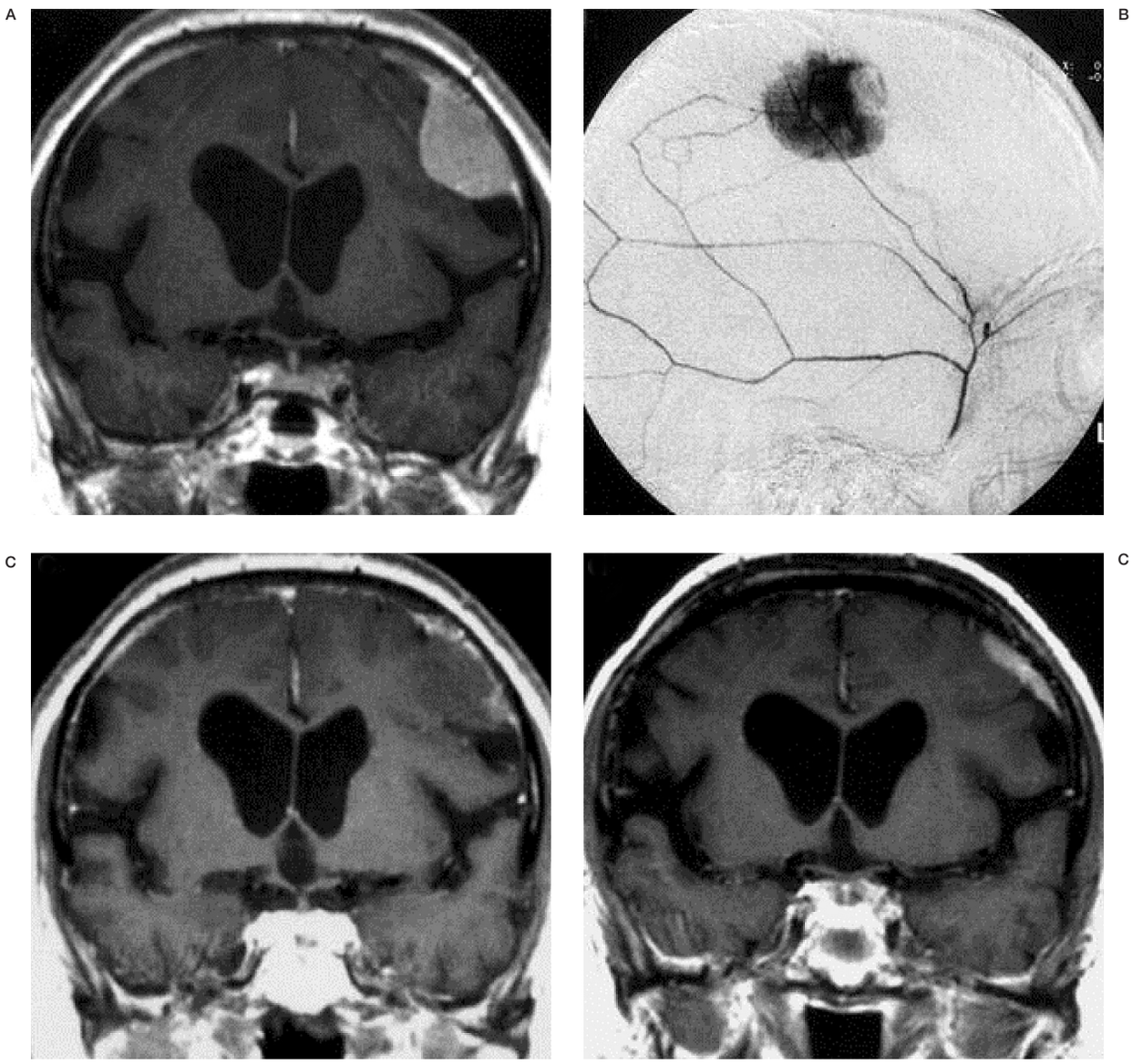

Figure 1 From Bendszus et Al:[48]. The patient is a 70-year-old man with symptoms of lumbar stenosis who could not be operated upon because of co-morbid disease. (A) Contrast enhanced T1-weighted MRI prior to embolization. (B) Angiography showed that the tumor was supplied exclusively by the middle meningeal artery, which was completely embolized. (C) Contrast enhanced T1-weighted MRI one day after embolization. (D) Contrast enhanced T1-weighted MRI at 23 month follow-up showing marked reduction of the tumor with minimal mass effect. (Permission granted for use)

Radiosurgery

Studies using radiosurgery as a primary therapy for small to medium sized meningiomas, in lieu of surgery, have recently appeared with increased frequency in the literature. The development of stereotactic methods has allowed the accurate delivery of focused radiation to tumors in fairly high doses, while minimizing the radiation exposure of adjacent brain tissue [4]. Actuarial control rates comparable to surgical resection have been reported for benign meningiomas using radiosurgery as the primary therapy – gamma-knife radiosurgery for 219 patients with meningiomas produced a 93% progression free survival rate at ten years [32], gamma-knife radiosurgery for 66 patients with parasaggital meningiomas produced a 93% progression free survival rate at five years [33], and stereotactic radiosurgery performed on 62

patients with meningiomas in multiple anatomic locations showed a 95% progression free survival rate at seven years [34].

However, like open surgery the complication rate associated with radiosurgery is fairly high. Reported complications related to radiosurgery of meningiomas have included death from hypothalamic dysfunction, stroke secondary to occlusion of the internal carotid artery and posterior carotid artery, symptomatic brain edema, cyst formation, and cranial nerve dysfunction. The rate of these complications has been reported between 2-16% [32-37]. This therapy is also limited as it is useful only for tumors three cm or less in size, and by the fact that it may take months to years to achieve improvement in symptoms [38]. Yet another concern with radiosurgery is that the long-term effects have yet to be fully established; there is concern about the possibility of radiation induced secondary tumors [39].

Meningioma embolization

The results from surgery and radiotherapy discussed above, dictate that new, safer, and potentially more efficacious therapies for meningiomas need to be developed. One promising new therapeutic approach is definitive embolization.

Meningiomas are generally highly vascular tumors that obtain their blood supply from the arteries of the adjacent dura and bone [7]. The techniques of interventional neuroradiology now allow superselective catherization of the arteries that supply a tumor [40]. When a catheter is positioned selectively in a feeder artery of a tumor, the artery can usually be safely embolized without injuring healthy tissue. Preoperative, trans-arterial embolization of meningiomas, first described by Manelfe et Al. in 1973 [41], is a procedure that is now widely performed at centers with neurointerventional capability [40]. This procedure has been shown to reduce intraoperative blood loss and, by softening the tumor, to increase the ease of surgical resection [42-46], although the utility of this procedure is matter of continuing controversy [47].

A number of reports have noted substantial necrosis and reduction in tumor volume following superselective embolization, with concomitant reductions in neurological symptoms [48-51]. This led some to consider using embolization as a definitive therapy for patients who are not good surgical candidates.

Bendszus et Al. treated seven such patients with embolization without subsequent surgery [48]. They used trisacryl gelatin microspheres (100-300 _m in diameter) to embolize external carotid feeders. They were able to achieve angiographic devascularization in the five patients whose tumors were supplied exclusively by the external carotid; the internal carotid fed part of the tumor in two patients and was not embolized. On early post-embolization MRI, four patients had no contrast enhancement other than a thin rim. Two had nodular enhancement in the part of the tumor supplied by the internal carotid artery. One patient continued to show intense contrast enhancement. Except for the patient with continued intense enhancement, substantial tumor shrinkage occurred and improvement in symptoms was noted. (Figure 1). With a mean follow-up of 20 months, the patients continued to be asymptomatic and tumor shrinkage continued (although the extent of tumor shrinkage was greatest in the first six months after embolization). Similarly, Koike et Al. published a case report of one patient embolized without subsequent surgery whose neurological symptoms resolved approximately ten days following embolization, and who remained nearly asymptomatic at a four year follow-up [50].

Potential advantages of definitive embolization

The data from Bendszus and Koike strongly suggest that interventional radiologists should conduct larger trials to confirm the result and to ensure the long-term efficacy of definitive embolization. If the effectiveness of definitive embolization is demonstrated there would be many potential advantages of using this therapeutic modality rather than surgery or radiotherapy. The rate of serious complications associated with embolization, particularly in the hands of a very experienced neurointerventionalist, is far lower than that of surgery; the post-procedure recovery time is much shorter for embolization (the patient, in most cases, can go home post-procedure day one) than it is for surgery. Further even if embolization provides only temporary control of the tumor and the patient must go on to surgery, there is potentially a significant advantage to be gained by temporarily controlling the tumor by non-operative means, as this may ultimately decrease the num-

ber of surgical interventions required for a given patient. The rate of re-operation for meningiomas is between 20-84% depending on the extent of initial resection [31]. Each re-operation is more technically challenging than the last, owing to post-operative changes. Decreasing the number of times a patient must undergo surgical resection provides a significant benefit, particularly in light of the significant morbidity and mortality associated with surgery. Additionally, definitive embolization may provide a treatment option for patients with surgically inaccessible tumors or whose general health does not permit the risks of surgery. Finally, treating the patient with endovascular therapy alone potentially spares the patient the psychological trauma associated with craniotomy.

Embolization also offers potential advantages when compared with radiosurgery. Radiosurgery is limited as it can only be used for meningiomas smaller than three cm in diameter, as it takes months to years to cause regression of symptoms, and as it has a substantial side effect profile (see the discussion above). Tumor embolization can treat tumors of all sizes, should result in improvement in symptoms approximately ten days after treatment [50], and likely has a lower incidence of side effects.

Potential Risks

If trials of definitive embolization are to be conducted, the risks of this experimental form of therapy must be considered. The major risks associated with definitive embolization include the risks of the embolization procedure and the risks associated with treating the tumor based on an imaging diagnosis alone.

Potential risks of intervention

The risks associated with embolization can conceptually be divided into those related to angiography and those related to the embolization of the tumor.

There are three classes of complications related to angiography that have been described – local (including groin haematoma and access site infection), systemic (including contrast induced renal failure and allergic reaction to contrast), and neurological. Cloft et Al. performed a meta-analysis of prospective studies (including a total of 3517 patients) examining the complication rates of cerebral angiography in patients with cerebral vascular disease processes [52]. The rate of permanent neurological complications was very low at 0.07%. The risk of serious non-neurological complications (which included cases of large haematoma, peripheral thromboembolic events, transient hypotension, transient hypertension, and infection) was also low at 0.6%. These serious non-neurological complications resulted in a permanent condition in only 0.3% of patients. Heiserman et Al. published a similar study of 1000 consecutive cerebral angiograms [53]. They found a 0.5% rate of permanent neurological complications related to angiography. However all of the complications were in cerebrovascular patients, a group for whom angiography presents greater risk than tumor patients. None of the 40 patients in the study who underwent angiography for tumors experienced permanent complications. These data, taken together, suggest a favorable safety profile for the angiography portion of the embolization procedure.

There are several case reports of serious complications related to meningioma embolization. These include neurological deficits from embolic material getting into the vessels supplying the cranial nerves and brain [54], ischemic necrosis of the scalp flap from embolization of scalp vessels, and various types of hemorrhage, including subarachnoid and intratumoral [45,54-56]. These major complications are, in most instances, due to breech in technique–either a failure to appreciate dangerous anastamoses or injecting the embolic material at pressures that are too high [54].

The long experience of the neurointerventional field with pre-operative embolization has shown it to be generally very safe. Probst et Al. reported that of 80 patients undergoing embolization only one developed a permanent neurological deficit (hypoesthesia in the sensory area of the third branch of the trigeminal nerve) [57]. Lasjaunias et Al., in a series of 185 patients, had one case of permanent cerebal deficit, two cases of permanent ophthalmic deficit, and five cases of transient facial nerve palsies [58]. Halbach et Al. reported that the institutional experience at UCSF involving several hundred cases of pre-operative meningioma embolizations had a complication rate of less than 0.5% [59]. Wakhloo et Al. in a series of 34 patients had one major complication (acute intratumoral bleeding following embolization which was successfully treated with emergent surgery,

leaving the patient neurologically intact), and three cases of transient neurological deficits following the procedure [51]. Richter et Al. in a series of 31 patients had three cases of post-embolization facial pain, one case of transient hemiplegia, and one case of thrombopenia (which was thought to probably be unrelated to the procedure) [42]. Chun et Al: in a series of 50 patients report only a single complication–a groin haematoma [44]. None of these studies had a procedure related death.

These data suggest that although the procedure is not without risks, embolization has a very favorable safety profile when compared with either surgery or radiosurgery. Further, it must be kept in mind when considering the risks of embolization that it is standard of care at many institutions for patients to undergo both embolization and subsequent surgery –such that most patients assume the risks of both of these interventions when undergoing standard treatment.

Risk of treating based on an imaging diagnosis alone.

One of the major risks that must be attended to in any protocol that proposes treating meningiomas with definitive embolization is the risk of misdiagnosis. Although surgery is undertaken based on radiological findings, the tumor tissue is retrieved during the procedure such that the diagnosis can be histologically confirmed, and subsequent treatments planned according to the findings. Definitive embolization will of course not allow for a tissue based diagnosis. One option to circumvent this limitation would be to perform a tumor biopsy prior to embolization. However, given the vascularity of meningiomas, this may add substantial risk to a definitive embolization protocol, such that the advantage of embolization in terms of safety is lost. Thus, it is important to consider the sensitivity and specificity of imaging at detecting benign meningiomas.

Benign meningiomas have a very characteristic appearance on both CT with contrast and MRI with gadolinium [60,61]. This, coupled with the fact that the great majority of extra-axial tumors are meningiomas, gives the radiographic diagnosis of meningioma a very high specificity. Nevertheless, there are several intracranial lesions that may rarely be mistaken for a benign meningioma. These include schwannomas, malignant meningiomas, brain metastasis (particularly from breast and prostate cancers), haemangiopericytoma, chordoma, chondrosacoma, central nervous tissue sarcoid [32,40], and tuberculosis. In the vast majority of instances these can be reliably differentiated from benign meningiomas as they have anatomic, morphologic, and other features not typically shared by meningiomas. Further specificity to the radiological findings can be added by considering the patients' history (i.e. slow onset and progression of symptoms, no history of other malignancy). Additionally, safeguards can be put in place to rule-out other diseases that may masquerade as meningiomas (such as prostate cancer with PSAs or breast cancer with mammograms).

Nevertheless, there is a small, but real risk, that patients may be misdiagnosed and thus improperly treated. This risk is somewhat mitigated by the fact that the safety of embolizing many of the tumors that might be mistaken for benign meningiomas, including schwannomas, malignant meningiomas, brain metastasis, and hemangiopericytoma, has been well documented [62-64]. Further, embolized patients can be followed very closely after the procedure for evidence of tumor progression/recurrence, so that patients with aggressive disease processes (such as malignant meningiomas, brain metastases, haemangiopericytoma, chondrosarcoma) can be readily detected early in the follow-up period and referred for appropriate treatment.

An approximation of the risk of misdiagnosis is provided in the study by Flickinger et Al. [32]. They followed 219 patients treated with gamma knife radiosurgery for imaging-diagnosed intracranial meningiomas (without pathologic diagnosis) for a mean follow-up of 29 months. Tumor progression occurred in seven of the patients, and in two of these progressors the initial diagnosis of meningioma was incorrect (one was a metastatic adenoid cystic carcinoma of the nasopharynx and the other was a chondrosarcoma). In one other patient, local control of the initial tumor was obtained, but the development of other brain metastases suggested that the initial diagnosis of meningioma was also incorrect. This gave them an actuarial rate of identifying a diagnosis other than meningioma of 2.3% at five years. It should be noted that patients in this study did not undergo angiography, as they would be in an embolization proto-

col. Benign meningiomas have a very characteristic appearance, with a "sunburst" pattern on angiography [40]. By adding this test to the diagnostic battery, it is likely that the rate of misdiagnosis in an embolization protocol will be lower than it was in this study that employed only MRI or CT.

It should be noted that the approach of treating meningiomas based on an imaging diagnosis alone has an extensive precedent in the radiosurgery literature [32,34-36], which should increase interventionalist comfort level with pursuing this approach.

Conclusions

While the current evidence for the safety and efficacy of definitive embolization is based solely on case reports and small series with limited follow-up and is therefore weak, the initial results are very promising. The limitations and dangers of surgery and radiosurgery dictate that neurointerventionalists pursue the development of this therapeutic approach. What are needed now are prospective trials that rigorously examine the safety and efficacy of definitive embolization.

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John Pile-Spellman, M.D.
Columbia University
177 Fort Washington Ave
MHB 8 SK
New York, NY 10032
(212) 305-6515

EDITORIAL COMMENT

Bateman et Al. examined the role of embolization as a unique treatment for intracranial meningiomas. This is an excellent review of outcomes in the treatment of meningiomas by surgery or radiosurgery. The authors are encouraged with the results of embolization as a definitive treatment for meningiomas. However, we must be cautious in our interpretation of these results since the two reports cited have a total of only eight patients and an average follow-up of twenty months.

Nevertheless, the proposal to consider prospective trials to determine the safety and efficacy of embolization as a unique therapy is an interesting one. This could include novel techniques in embolization with new embolic agents and the use of chemotherapeutic drugs. Combined suites, with both MR and angiography capabilities, may facilitate a "real time" assessment of the effects of the embolic agents. Bateman et Al. has thrown down the guantlet for the interventional neuroradiology community to consider.

Dr Robert A. Willinsky
Professor of Medical Imaging
The Toronto Western Hospital, University,
Health Network 399 Bathurst St.
Toronto, ON, Canada M5T 2S8
E-mail: robert.willinsky@uhn.on.ca
www.brainavm.com

EDITORIAL COMMENT

Authors reviewed number of published articles regarding treatment of intracranial meningiomas including surgery, radiosurgery and embolization. They concentrated in discussion of mortality and morbidity following each method of treatment. Many publications were referred, suggesting higher mortality rates, compared to other modes of treatment, radiosurgery and embolization. Indications and results including morbidity of radiosurgery were also discussed. Concerning embolization as a sole mode of treatment, authors made references to two reports, one with seven cases and one with single case. The first article reported that four out of five cases with exclusive dural supply to tumor showed shrinkage after embolization in mean 20 months follow-ups. The second one is a case report of meningioma embolized and followed for four years clinically. Authors suggest a prospective trial to examine the safety and efficacy of definitive embolization based on comparison of these reports to published surgical results.

It is difficult to accept authors' argument suggesting a clinical trial of treating intracranial meningiomas on the basis of mortality and morbidity alone. Besides, cases included in surgery reports are in hundreds, but embolization cases referred are only five. As cases illustrated in the reports, proper embolization can induce tumor necrosis and eventual stable shrinkage of mass. It can only be achieved by deep penetration of embolic material into the tumor bed of which is supplied exclusively by dural arteries.

Meningiomas occur in any part of intracranial space, including ventricles. Depending upon its location and anatomical predisposition as well as vascular arrangement, its blood supply is determined accordingly. Even for similar size and location, some may have blood supply only from meningeal arteries, however, others may have supply not only from meningeal arteries but from pial arteries. In fact, majority of symptomatic meningiomas have pial arterial supply in varying degree. In some locations, major supply may be from the pial arteries, either internal carotid or vertebrobasilar arteries which can not be embolized so safely.

These factors are important to consider, and deserve to be mentioned after this review. As commented previously, it is possible to stabilize certain size meningiomas in certain locations. It is not feasible to apply embolization as definitive treatment to all meningiomas.

In Sup Choi MD
Lahey Clinic Medical Center
Burlington, USA